platelet aggregation inhibitor, is indicated in high risk cardiovascular patients to prevent recurrent ischemic events. Therefore, the purpose of this study was to determine the safety and efficacy of clopidogrel administration initiated in the early postoperative period after OPCAB.
Methods: 30-day follow-up of 364 OPCAB patients from Jan-June, 2001 was determined from prospectively collected data. 193 patients received clopidogrel approximately 4 hours postoperatively if chest tube output < 100cc/hr for 4 hours and then daily for 4-6 weeks. Telephone follow-up was made 6 – 12 months after OPCAB in 93% of clopidogrel and 88% of non-clopidogrel patients. Adverse cardiovascular events were defined as unstable angina, myocardial infarction, transient ischemic attack, or stroke.
Results: The 193 clopidogrel patients had significantly higher preoperative risk scores compared to the 171 non-clopidogrel patients: 3-vessel CAD, 62% vs. 50%, prior PTCA, 26% vs. 16%, prior intracoronary stent placement 19% vs. 7%, and CCS angina III-IV, 58% vs. 37%, p<0.02. None of the clopidogrel patients required reoperation for mediastinal hemorrhage. No group differences in mortality were observed at 6 months. For all risk categories, no differences in adverse cardiovascular events were observed, 3.4% of clopidogrel patients vs. 5.3% for non-clopidogrel patients, p=NS. However, in low risk patients (CCS angina score <2 and 1-2 vessel CAD), fewer adverse cardiovascular events occurred in the clopidogrel group, 1/92 (1.1%) vs. 6/73 (8.2%), p=0.045. Gastrointestinal bleeding occurred in 2.2% clopidogrel patients vs. 0.7% non-clopidogrel patients, p=NS.
Conclusions: According to this protocol, OPCAB patients can safely receive clopidogrel in the early postoperative period without increased risk for mediastinal hemorrhage. Early clopidogrel administration after OPCAB may be associated with a reduction in short-term adverse cardiovascular events.

Noon

1059-16 Can Autologous Myoblast Transplantation Decrease Chronic Ischemic Mitral Regurgitation?

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Background: Extensive work has confirmed the relationship between ischemic mitral regurgitation (MR) and remodeling of the infarcted left ventricle (LV), which displaces apically the papillary muscles to which the mitral leaflets are anchored. This study was thus designed to assess whether transplantation (Tx) of skeletal myoblasts could reduce mitral leaflet tethering and chronic ischemic MR by decreasing endsystolic volumes (ESV) and reshaping the infarcted wall. **Methods:** An open-chest infero-basal infarction was created in 14 sheep, thereby resulting in a MR which progressively developed over the ensuing 8 weeks. At this time point, sheep were randomly allocated to receive previously expanded autologous skeletal myoblasts (230 million cells, of which 65% were myoblasts identified by a positive staining for CD56, n = 7) or to serve as controls receiving culture medium only (n = 7). All injections were made in multiple sites in the infarcted posterior bulging wall. 3D echocardiography was used for serial evaluations performed immediately after infarction, 2 months therafter (preTx) and 2 additional months after Tx. End points included blinded measurements of the tethering distance between the ischemic medial papillary muscle tip and the anterior annulus, LV ejection fraction and stroke volume while 2D echo was used for assessing wall motion score (WMS). **Results:** Parameters (mean ± SEM) were similar at baseline and at 2 months between the 2 groups. Two months after Tx (i.e., 4 months post infarction), myoblast Tx was found to have reduced the progression of ischemic MR (regurgitation volume: 0.7 ± 0.5 *vs.* 5.7 ± 0.9 mL in controls, $p < 0.01$), the increase in ESV (30.4 ± 1.2 mL *vs.* 42.0 ± 2.8 mL in controls, $p < 0.01$) and the tethering distance (0.01 ± 0.05 cm *vs.* 0.44 ± 0.12 cm in controls, $p = 0.01$). The benefits of myoblast Tx were also evidenced by a lesser decrease of LV ejection fraction (2.9 ± 1.8% *vs.* 7.8 ± 1.9% in controls, $p < 0.01$) and a significant improvement of the WMS (-0.6 ± 0.03 *vs.* 0.1 ± 0.01, respectively, $p < 0.01$). **Conclusion:** Tx of skeletal myoblasts may attenuate mild to moderate chronic ischemic MR by decreasing ESV and reshaping the infarcted LV wall, thereby enhancing valve coaptation.

Noon

1059-17 Prolongation of Atrial Effective Refractory Period by Biatrial Subthreshold Stimulation

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Timely subthreshold atrial extrastimuli (AE) delivered in the refractory period can prevent the induction of atrial fibrillation (AF) by another AE delivered in the vulnerable period. This study aims to investigate the effect of subthreshold stimulation (SS) on atrial effective refractory period (AERP).
Methods: 14 patients without history of AF (9 male and 5 female, mean age of 51) were recruited. AERP of right atrial septum was determined after 5 minutes of pacing with 4 different protocols and 3 sets of cycle length (CL) commenced in random order with 10 minutes of washout time between each protocol. Protocols were as shown in table below, pacing at high right atrium (HRA) and bi-atrial (HRA + coronary sinus) was at twice diastolic threshold. SS was commenced by introduction of an electrical impulse of 2.0msec in duration and 20mA in amplitude at 50msec after the preceding captured pacing impulse. The mean of 3 AERP measurements was recorded.
Results: Analysis showed that the pacing protocol but not the CL had significant effect on AERP. Multiple comparisons showed that the Bi-atrial + SS group had significant longer AERP than all other groups (protocol 4 versus protocol 1, p< 0.001; versus protocol 2, p< 0.021; versus protocol 3, p< 0.002)

AERP±SD(msec)/ CL(msec)	HRA	HRA + SS	Bi-atrial	Bi-atrial +SS
600	218.2 ± 20.0	221.8 ± 20.8	216.8 ± 20.7	242.1 ± 31.8
500	218.6 ± 20.0	224.3 ± 22.2	220.0 ± 24.5	237.9 ± 30.7
400	211.1 ± 24.0	226.8 ± 20.0	225.4 ± 25.5	240.0 ± 32.3

Conclusion: Bi-atrial SS significantly prolonged AERP. It is plausible that because SS is relatively localized, the larger the area of atrium receives SS the greater its effect on AERP. The study result suggests that bi-atrial SS may be an effective therapy for prevention of AF.

Noon

1059-18 The Role of Slow Delayed Rectifier Potassium Current (I_{Ks}) in Cardiac Electrophysiology and Atrial Fibrillation

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Background: The role of I_{Ks} in cardiac electrophysiology has been controversial: direct experimental analysis has been limited by a lack of adequate probes. In this study, we used a highly-selective I_{Ks} blocker, HMR1556 (HMR), to evaluate the I_{Ks} contribution to cardiac electrophysiological function and AF maintenance.
Methods: Anesthetized open-chest dogs were studied before and after HMR alone (1 mg/kg IV), HMR plus nadolol 0.5 mg/kg IV, dofetilide alone (D, 0.16 mg/kg IV) or HMR plus D (H/D).
Results: HMR alone increased atrial (AERP at cycle length, CL, 300 ms, $AERP_{300}$: 88±11, M±SD, to 105±13 ms, *p<.05) and ventricular ($VERP_{600}$: 154±14 to 190±32 ms*) effective refractory period with positive or no frequency-dependence, and increased sinus node recovery time (SNRT). Beta blockade eliminated HMR effects on AERP ($AERP_{300}$: 99±5 to 101±17 ms) and SNRT, but did not alter changes in VERP ($VERP_{600}$: 177±19 to 211±11 ms*). D prolonged both AERP ($AERP_{300}$: 92±4 to 143±17 ms*) and VERP ($VERP_{600}$: 179±18 to 213±29 ms*) with reverse use dependence. In the presence of D, HMR effects were significantly increased, e.g. HMR increased $AERP_{300}$ 22±10% vs control, H/D increased $AERP_{300}$ 29±12%* vs D; HMR increased $VERP_{600}$ 24±19% vs control, H/D increased $VERP_{600}$ 32±5%* vs D. In addition, Wenckebach CL was not affected by HMR alone, but was greatly increased by H/D vs D alone. HMR shortened the duration of induced vagotonic AF (DAF, 1077±198 to 471±339 sec*), an effect abolished by β-blockade. D had no significant effect on DAF (916±216 to 732±243 ms), but H/D markedly decreased DAF (to 77±73 ms, p<.001).
Conclusions: I_{Ks} plays a role in baseline atrial, ventricular and SA node repolarization in vivo, with atrial and SA nodal effects being dependent on background β-adrenoceptor stimulation. I_{Ks} also plays a role in AF maintenance in the presence of intact sympathetic tone. I_{Ks} effects are particularly important in the presence of reduced repolarization reserve, as indicated by the synergistic interaction between HMR and D. This constitutes the first evaluation of the role of I_{Ks} for in vivo cardiac electrophysiology and highlights the distinct profile of I_{Ks} vs I_{Kr} blockade.

Noon

1059-19 Effect of Cardiac Resynchronization Therapy in Patients With Right Bundle Branch Block

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Background: Cardiac resynchronization therapy (CRT) is indicated for pts with heart failure and QRS delay. There is no published data evaluating CRT in pts with right bundle branch block (RBBB). Methods: Data was analyzed at baseline and 6 months in the 501 pts with NYHA FC II-IV heart failure and ICD indications enrolled in the CONTAK CD Trial (Guidant, Corp). Results: A total of 66/501 (13%) pts had RBBB, 271/501 (62%) had LBBB and 164/501 (38%) had IVCD. There were no differences in baseline characteristics between RBBB and non-RBBB pts. Unlike non-RBBB pts, RBBB pts did not demonstrate improvement in symptom status, heart size or LVEF (Table). NYHA FC did not improve with RBBB pts and the subset of enrolled pts with FC III-IV symptoms also showed no benefit. Conclusion: CRT may not benefit RBBB pts. A meta-analysis of the completed controlled trials of RBBB pts is needed to corroborate these findings. Alternative stimulation sites should be tested in this subset.

Table

Endpoint	Time	RBBB CRT/No CRT, p	Non RBBB CRT/No CRT, p
V02 (ml/kg/min)	Baseline 6 mo change	13±.5/13±.5 -1±.6/-.1±.6, ns	14±.2/14±.2 1±.3/0±.3, .009
6 min walk (m)	Baseline 6 mo change	302±14/302±13 10±18/21±17, ns	320±6/320±6 39±8/13±8, .02
LVIDs (sys/mm)	Baseline 6 mo change	54±1/54±1 -1±2/-2±2, ns	59±.5/59±.5 59±.5/59±.5 -4±.7/-.6±.7, < .001
LVEF (%)	Baseline 6 mo change	31±1/31±1 2±2/4±1, ns	27±.4/27±.4 6±.8/3±.8, .008